A. Harris
627694

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=> fil reg
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.60       0.60

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for details.

=> e gp75/cn 5
E1            1     GP63 SURFACE PROTEASE (LEISHMANIA INFANTUM)/CN
E2            1     GP64 GLYCOPROTEIN (POLYSPHONDYLIUM PALLIDUM CLONE C-P644
PRE
                    CURSOR)/CN
E3            0 --> GP75/CN
E4            2     GPA/CN
E5            1     GPA (AMIDE)/CN

=> fil medl,caplus,biosis,embase,wpids,jicst,scisearch
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.31       0.91

FILE 'MEDLINE' ENTERED AT 15:08:37 ON 23 APR 2001

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Prepared by M. Hale 308-4258

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=> s (non human or insect)(2a)cell line and (human differ? antigen or human melanocyte? or gp75 or gp 75)
L1 0 FILE MEDLINE
L2 0 FILE CAPLUS
L3 0 FILE BIOSIS
L4 0 FILE EMBASE
L5 1 FILE WPIDS
L6 0 FILE JICST-EPLUS
L7 0 FILE SCISEARCH

TOTAL FOR ALL FILES
L8 1 (NON HUMAN OR INSECT)(2A) CELL LINE AND (HUMAN DIFFER? ANTIGEN OR HUMAN MELANOCYTE? OR GP75 OR GP 75)

=> d

L8 ANSWER 1 OF 1 WPIDS COPYRIGHT 2001 DERWENT INFORMATION LTD
AN 1998-348236 [30] WPIDS
DNC C1998-107615
TI Stimulating immune response to differentiation antigen - which is expressed by tumour, comprises use of altered differentiation antigen expressed in, or derived from, another species.
DC B04 D16
IN HOUGHTON, A; NAFTZGER, C; VIJAYASARADHI, S
PA (SLOK) SLOAN KETTERING INST CANCER RES
CYC 21
PI WO 9825574 A2 19980618 (199830)* EN 19p A61K000-00
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE
W: CA JP US
EP 959893 A2 19991201 (200001) EN A61K035-12
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE
ADT WO 9825574 A2 WO 1997-US22669 19971210; EP 959893 A2 EP 1997-954555 19971210, WO 1997-US22669 19971210
FDT EP 959893 A2 Based on WO 9825574
PRAI US 1997-36419 19970217; US 1996-32535 19961210
IC ICM A61K000-00; A61K035-12
ICS A61K035-56; C12N005-06; C12N005-10; C12N005-18; C12N005-22; C12N015-06; C12N015-12; C12N015-63; C12N015-85

=> s immun? respons? and different? antigen? and antigen?
L9 313 FILE MEDLINE
L10 231 FILE CAPLUS
L11 348 FILE BIOSIS
L12 376 FILE EMBASE
L13 24 FILE WPIDS
L14 237 FILE JICST-EPLUS
L15 294 FILE SCISEARCH

TOTAL FOR ALL FILES
L16 1823 IMMUN? RESPONS? AND DIFFERENT? ANTIGEN? AND ANTIGEN?

=> s (gp75 or gp 75) and l16

L17 4 FILE MEDLINE
L18 6 FILE CAPLUS
L19 6 FILE BIOSIS
L20 5 FILE EMBASE
L21 0 FILE WPIDS
L22 0 FILE JICST-EPLUS
L23 5 FILE SCISEARCH

TOTAL FOR ALL FILES
L24 26 (GP75 OR GP 75) AND L16

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L25 4 FILE MEDLINE
L26 6 FILE CAPLUS
L27 6 FILE BIOSIS
L28 5 FILE EMBASE
L29 0 FILE WPIDS
L30 0 FILE JICST-EPLUS
L31 5 FILE SCISEARCH

TOTAL FOR ALL FILES
L32 26 L24 NOT L8

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PROCESSING COMPLETED FOR L32
L33 7 DUP REM L32 (19 DUPLICATES REMOVED)

=> d 1-7 cbib abs;s houghton a?/au,in;s naftger c?/au,in;s sataluri v?/au,in;s gregor p?/au,in

L33 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2001 ACS
1998:402278 Document No. 129:66831 Method and compositions for stimulation of an **immune response** to a **differentiation antigen** stimulated by an altered **differentiation antigen**. Houghton, Alan; Naftzger, Clarissa; Vijayasaradhi, Setaluri (Sloan-Kettering Institute for Cancer Research, USA; Houghton, Alan; Naftzger, Clarissa; Vijayasaradhi, Setaluri). PCT Int. Appl. WO 9825574 A2 19980618, 19 pp. DESIGNATED STATES: W: CA, JP, US; RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE. (English). CODEN: PIXXD2. APPLICATION: WO 1997-US22669 19971210. PRIORITY: US 1996-32535 19961210; US 1997-36419 19970217.
AB Tolerance of the immune system for self-**differentiation antigens** can be overcome and an **immune response** stimulated by administration of a therapeutic **differentiation antigen**. The therapeutic **differentiation antigen** is altered with respect to the target **differentiation antigen** in the individual being treated (i.e., the **differentiation antigen** to which an **immune response** is desired) in one of three ways. First, the therapeutic **differentiation antigen** may be syngeneic with the target **differentiation antigen**, provided that therapeutic **differentiation antigen** is expressed in cells of a species different from the individual being treated. For example, a human **differentiation antigen** expressed in insect or other non-human host cells can be used to stimulate

an **immune response** to the **differentiation antigen** in a human subject. Second, the therapeutic **differentiation antigen** may be a mutant form of a syngeneic **differentiation antigen**, for example a glycosylation mutant. Third, the therapeutic **differentiation antigen** may be a **differentiation antigen** (wild-type or mutant) of the same type from a species different from the individual being treated. For example, a mouse **differentiation antigen** can be used to stimulate an **immune response** to the corresponding **differentiation antigen** in a human subject. Administration of altered **antigens** in accordance with the invention results in an effective immunity against the original **antigen** expressed by the cancer in the treated individual.

L33 ANSWER 2 OF 7 MEDLINE DUPLICATE 1
1999031212 Document Number: 99031212. PubMed ID: 9813669.
Adenovirus-mediated expression of melanoma **antigen gp75** as immunotherapy for metastatic melanoma. Hirschowitz E A; Leonard S; Song W; Ferris B; Leopold P L; Lewis J J; Bowne W B; Wang S; Houghton A N; Crystal R G. (Division of Pulmonary and Critical Care Medicine, New York Hospital-Cornell Medical Center, NY 10021, USA.) GENE THERAPY, (1998 Jul) 5 (7) 975-83. Journal code: CCE; 9421525. ISSN: 0969-7128. Pub. country: ENGLAND: United Kingdom. Language: English.

AB Melanocyte **differentiation antigens**, such as the brown locus protein **gp75**, are potential biological targets for immunotherapy. We investigated whether expression of the murine **gp75** cDNA mediated by an adenovirus (Ad) vector could induce melanoma rejection using this model self **antigen** that usually induces tolerance, and whether Ad vector-directed production of interleukin-2 (IL2) might augment this response. To evaluate this approach, Ad vectors were constructed containing the murine **gp75** cDNA (Ad.**gp75**) and the human IL2 cDNA (Ad.IL2). Efficacy was evaluated in C57BI/6 mice challenged i.v. with 10(5) B16 cells, using the number of lung metastases as the efficacy parameter. Naive control mice developed 175 +/- 12 metastases by day 14. Controls receiving intranasal Ad.IL2 1 day after B16 cell injection, intraperitoneal (i.p.) mitomycin-C-treated B16 cells +/- i.p. Ad.IL2 before B16 cell challenge and Ad.beta gal-treated mice had similar numbers of metastases as controls (P > 0.1). In marked contrast, preimmunization with intradermal Ad. **gp75** provided dramatic reduction in the number of lung metastases (52 +/- 7, 29% of control). Addition of regional (intranasal delivery to the lung) Ad.IL2 to intradermal Ad.**gp75** preimmunization 1 day following tumor challenge provided further protection (18 +/- 6, 10% of control). Depletion of CD4+ and CD8+ T-cell subsets effectively blocked the protective effect seen following immunization. Adoptive transfer of macrophage-depleted splenocytes from Ad.**gp75**-immunized mice similarly afforded significant protection against B16 tumor cell challenge. Further, serum obtained 21 days following Ad.**gp75** immunization showed no detectable anti-**gp75** antibody by immunoprecipitation. These results suggest that immunization with Ad. **gp75** induces cellular **immune responses** that

are capable of rejecting B16 melanoma in a host that is usually tolerant to **gp75 antigen**.

L33 ANSWER 3 OF 7 MEDLINE DUPLICATE 2
1998118567 Document Number: 98118567. PubMed ID: 9435247. Fc receptors are required in passive and active immunity to melanoma. Clynes R; Takechi Y; Moroi Y; Houghton A; Ravetch J V. (Laboratory of Molecular Genetics and Immunology, Rockefeller University, New York, NY, USA.. clynesr@rockvax.rockefeller.edu) . PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1998 Jan 20) 95 (2) 652-6. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States. Language: English.

AB Effective tumor immunity requires recognition of tumor cells coupled with the activation of host effector responses. Fc receptor (FcR) gamma-/- mice, which lack the activating Fc gamma R types I and III, did not demonstrate protective tumor immunity in models of passive and active immunization against a relevant tumor **differentiation antigen**, the brown locus protein **gp75**. In wild-type mice, passive immunization with mAb against **gp75** or active immunization against **gp75** prevented the development of lung metastases. This protective response was completely abolished in FcR gamma-deficient mice. **Immune responses** were intact in gamma-/- mice because IgG titers against **gp75** develop normally in gamma-/- mice immunized with **gp75**. However, uncoupling of the Fc gamma R effector pathway from antibody recognition of tumor **antigens** resulted in a loss of protection against tumor challenge. These data demonstrate an unexpected and critical role for FcRs in mediating tumor cytotoxicity in vivo and suggest that enhancement of Fc gamma R-mediated antibody-dependent cellular cytotoxicity by inflammatory cells is a key step in the development of effective tumor immunotherapeutics.

L33 ANSWER 4 OF 7 BIOSIS COPYRIGHT 2001 BIOSIS
1997:331723 Document No.: PREV199799630926. **Immune response** to melanosomal **differentiation antigen** induced by altered **antigen**. Bartido, Shirley M.; Hara, Isao; Naftzger, Clarissa; Wang, Sigun; Weber, Lawrence; Yang, George; Xu, Yiqing; Setaluri, Vijayasaradhi; Qin, Jie; Moroi, Yoichi; Houghton, Alan N.. Memorial Sloan-Kettering Cancer Cent., New York, NY USA. Pigment Cell Research, (1997) Vol. 10, No. 1-2, pp. 103. Meeting Info.: XVIth International Pigment Cell Conference Anaheim, California, USA October 29-November 1, 1996 ISSN: 0893-5785. Language: English.

L33 ANSWER 5 OF 7 MEDLINE DUPLICATE 3
97121471 Document Number: 97121471. PubMed ID: 8962137. **Immune response** to a **differentiation antigen** induced by altered **antigen**: a study of tumor rejection and autoimmunity. Naftzger C; Takechi Y; Kohda H; Hara I; Vijayasaradhi S; Houghton A N. (Swim Across America Laboratory, Memorial Sloan-Kettering Cancer Center, New York, NY 10021, USA.) PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1996 Dec 10) 93 (25) 14809-14. Journal code: PV3; 7505876. ISSN: 0027-8424. Pub. country: United States.

Language: English.

AB Recognition of self is emerging as a theme for the immune recognition of human cancer. One question is whether the immune system can actively respond to normal tissue autoantigens expressed by cancer cells. A second but related question is whether immune recognition of tissue autoantigens can actually induce tumor rejection. To address these issues, a mouse model was developed to investigate **immune responses** to a melanocyte **differentiation antigen**, tyrosinase-related protein 1 (or **gp75**), which is the product of the brown locus. In mice, immunization with purified syngeneic **gp75** or syngeneic cells expressing **gp75** failed to elicit antibody or cytotoxic T-cell responses to **gp75**, even when different immune adjuvants and cytokines were included. However, immunization with altered sources of **gp75 antigen**, in the form of either syngeneic **gp75** expressed in insect cells or human **gp75**, elicited autoantibodies to **gp75**. Immunized mice rejected metastatic melanomas and developed patchy depigmentation in their coats. These studies support a model of tolerance maintained to a melanocyte **differentiation antigen** where tolerance can be broken by presenting sources of altered **antigen** (e.g., homologous xenogeneic protein or protein expressed in insect cells). **Immune responses** induced with these sources of altered **antigen** reacted with various processed forms of native, syngeneic protein and could induce both tumor rejection and autoimmunity.

L33 ANSWER 6 OF 7 MEDLINE DUPLICATE 4

96042160 Document Number: 96042160. PubMed ID: 7595233. Implicating a role for immune recognition of self in tumor rejection: passive immunization against the brown locus protein. Hara I; Takechi Y; Houghton A N. (Memorial Sloan-Kettering Cancer Center, New York 10021, USA.) JOURNAL OF EXPERIMENTAL MEDICINE, (1995 Nov 1) 182 (5) 1609-14. Journal code: I2V; 2985109R. ISSN: 0022-1007. Pub. country: United States. Language: English.

AB The immune system can recognize **differentiation antigens** that are selectively expressed on malignant cells and their normal cell counterparts. However, it is uncertain whether immunity to **differentiation antigens** can effectively lead to tumor rejection. The mouse brown locus protein, **gp75** or tyrosinase-related protein 1, is a melanocyte **differentiation antigen** expressed by melanomas and normal melanocytes. The **gp75 antigen** is recognized by autoantibodies and autoreactive T cells in persons with melanoma. To model autoimmunity against a melanocyte **differentiation antigen**, mouse antibodies against **gp75** were passively transferred into tumor-bearing mice. Passive immunization with a mouse monoclonal antibody against **gp75** induced protection and rejection of both subcutaneous tumors and lung metastases in syngeneic C57BL/6 mice, including established tumors. Passive immunity produced coat color alterations but only in regenerating hairs. This system provides a model for autoimmune vitiligo and shows that **immune responses** to melanocyte **differentiation antigens** can influence mouse coat color. Immune recognition of a melanocyte **differentiation antigen** can reject tumors, providing a basis for targeting tissue autoantigens expressed on cancer.

L33 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2001 ACS DUPLICATE 5
1994:215142 Document No. 120:215142 Differential recognition of sequences within the encephalitogenic region of myelin basic protein capable of eliciting cell-mediated **immune responses** in experimental autoimmune encephalomyelitis. Malotky, Michele K.H.; Paterson, Philip Y.; Miller, Stephen D. (Med. Sch., Northwestern Univ., Chicago, IL, 60611, USA). J. Neuroimmunol., 48(2), 135-42 (English) 1993.
CODEN: JNRIDW. ISSN: 0165-5728.

AB The fine specificity of myelin basic protein (MBP) epitopes capable of eliciting in vivo delayed-type hypersensitivity responses in Lewis rats with exptl. autoimmune encephalomyelitis (EAE) was compared to those eliciting in vitro **antigen**-specific T cell proliferation and augmentation of disease transfer. Utilizing a panel of synthetic peptides with sequences representing the 68-86 region of guinea pig (GP-) or bovine myelin basic protein (B-MBP), animals were primed with one species of peptide and subsequently challenged with either the same peptide or peptides with truncations or substitutions representative of the other species of MBP. In regard to minimal length sequences capable of eliciting delayed-type hypersensitivity (DTH), rats primed with GP-MBP and complete Freund's adjuvant (CFA) exhibited a hierarchical pattern of responsiveness to challenge with a series of truncated peptides, ranking as follows: GP-68-86 > GP-72-86 > GP-68-84 .mchgt. **GP-75**-86 = no activity. This response pattern corresponds to that previously reported for T cell proliferation and activation for disease transfer. A comparison of these T cell-mediated immune parameters, as elicited by the substituted peptides, revealed the response patterns of DTH reactivity to be similar to that previously described for in vitro T cell proliferation with significant DTH responses generated only by the peptide species for which the animal was primed. In contrast, a cross-reactive pattern of recognition was obsd. in cells mediating disease transfer, with all four 68-86 sequences capable of augmenting activation for adoptive transfer of disease, regardless of the peptide species for which the animal was primed. The **differential antigen** recognition patterns obsd. for these EAE-assocd. **immune responses** supports the hypothesis that multiple TH cell subsets are involved in disease pathogenesis.

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L34 262 FILE MEDLINE
L35 203 FILE CAPLUS
L36 332 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L37 245 FILE EMBASE
L38 34 FILE WPIDS
L39 1 FILE JICST-EPLUS
'IN' IS NOT A VALID FIELD CODE
L40 434 FILE SCISEARCH

TOTAL FOR ALL FILES

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L41       1511 HOUGHTON A?/AU,IN


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L44          0 FILE BIOSIS
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L47          0 FILE JICST-EPLUS
'IN' IS NOT A VALID FIELD CODE
L48          0 FILE SCISEARCH

TOTAL FOR ALL FILES
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L56          0 FILE SCISEARCH

TOTAL FOR ALL FILES
L57          0 SATALURI V?/AU,IN


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L62         31 FILE WPIDS
L63          2 FILE JICST-EPLUS
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L64         93 FILE SCISEARCH

TOTAL FOR ALL FILES
L65        514 GREGOR P?/AU,IN

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L68          1 FILE BIOSIS
L69          1 FILE EMBASE
L70          0 FILE WPIDS
L71          0 FILE JICST-EPLUS
L72          2 FILE SCISEARCH
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TOTAL FOR ALL FILES
L73 6 L65 AND L41

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PROCESSING COMPLETED FOR L73
L74 2 DUP REM L73 (4 DUPLICATES REMOVED)

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L74 ANSWER 1 OF 2 SCISEARCH COPYRIGHT 2001 ISI (R)
2001:75194 The Genuine Article (R) Number: 372WB. Measurement by Elispot of interferon-gamma secreting T cells before and after T cell depletion in donor bone marrow grafts.. Schaed S G (Reprint); Alpdogan O; Trcka J; **Gregor P D**; **Houghton A N**; O'Reilly R J; Collins N H. Mem Sloan Kettering Canc Ctr, New York, NY 10021 USA. BLOOD (16 NOV 2000) Vol. 96, No. 11, Part 1, pp. 183A-183A. MA 786. Publisher: AMER SOC HEMATOLOGY. 1900 M STREET. NW SUITE 200, WASHINGTON, DC 20036 USA. ISSN: 0006-4971. Pub. country: USA. Language: English.

L74 ANSWER 2 OF 2 MEDLINE DUPLICATE 1
2000306559 Document Number: 20306559. PubMed ID: 10850386. Injection of DNA encoding granulocyte-macrophage colony-stimulating factor recruits dendritic cells for immune adjuvant effects. Bowne W B; Wolchok J D; Hawkins W G; Srinivasan R; **Gregor P**; Blachere N E; Moroi Y; Engelhorn M E; **Houghton A N**; Lewis J J. (Memorial Sloan-Kettering Cancer Center, New York, NY 10021, USA.) CYTOKINES, CELLULAR AND MOLECULAR THERAPY, (1999 Dec) 5 (4) 217-25. Journal code: CUS; 9713367. ISSN: 1368-4736. Pub. country: ENGLAND: United Kingdom. Language: English.

AB An important issue for effective vaccines is the development of potent adjuvants that can facilitate induction or augmentation of immunity. Granulocyte-macrophage colony-stimulating factor (GM-CSF) is a growth factor for myeloid progenitors of monocytes and dendritic cells (DC), which upon maturation are antigen-presenting cells (APC). The adjuvant effects of inoculation of DNA encoding GM-CSF into skin were studied. Initial experiments examined whether the GM-CSF gene injected into the skin of mice could affect the density of epidermal DC (Langerhans cells). DNA encoding GM-CSF delivered by particle bombardment into skin resulted in a significant increase of epidermal DC at the inoculation site. Kinetic analysis of epidermal recruitment after GM-CSF inoculation showed an increase in DC that peaked at seven days. This increase was accompanied by recruitment of DC into draining lymph nodes. The adjuvant effects of DNA encoding GM-CSF inoculated into skin were measured by the ability to augment antibody and T-cell responses against poorly immunogenic tumor antigens. Peptide immunization at skin sites containing epidermal DC newly recruited by GM-CSF DNA elicited T-cell responses against mutant p53, whereas peptide immunization of control skin sites did not elicit any detectable T-cell responses. Likewise, generation of antibodies following immunization with DNA encoding human gp75TRP1, a tyrosinase family member

expressed by melanomas, was accelerated and protection from tumor challenge augmented by GM-CSF DNA.

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L77           9 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L78           5 FILE EMBASE
L79           1 FILE WPIDS
L80           0 FILE JICST-EPLUS
'IN' IS NOT A VALID FIELD CODE
L81           4 FILE SCISEARCH

TOTAL FOR ALL FILES
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L85           0 FILE BIOSIS
'IN' IS NOT A VALID FIELD CODE
L86           0 FILE EMBASE
L87           0 FILE WPIDS
L88           0 FILE JICST-EPLUS
'IN' IS NOT A VALID FIELD CODE
L89           0 FILE SCISEARCH

TOTAL FOR ALL FILES
L90           0 SATALURI V?/AU,IN

=> s l82 and l65 and l41
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L93           0 FILE BIOSIS
L94           0 FILE EMBASE
L95           0 FILE WPIDS
L96           0 FILE JICST-EPLUS
L97           0 FILE SCISEARCH

TOTAL FOR ALL FILES
L98           0 L82 AND L65 AND L41

=> del his y

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COST IN U.S. DOLLARS                          SINCE FILE      TOTAL
                                                 ENTRY    SESSION
FULL ESTIMATED COST                              78.27      79.18

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)    SINCE FILE      TOTAL
                                                 ENTRY    SESSION
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